case can be completely predicated upon the blood-chemical findings, but we do insist upon the great value of this work in this and allied conditions. The determination of the amount of creatinin in these cases has given us some figures which seem to bear out the contention of Myers and Lough[10] that a percentage of creatinin that exceeds 5 mgm. per 100 c.c. of blood bodes ill for the patient. It will be noted that in the cases of O'Connor, Fischer, and Ship we had a creatinin value around or above 5 mgm. These three cases died. In the case of Ship at the time his blood was taken his clinical condition did not seem to be nearly as bad as that of Huth, yet the creatinin value of the former was above the so-called fatal point, whereas the latter had only a creatinin retention of 3.83 mgm. The of case Ship, however, vindicated the prognostic value of a high creatinin retention, as he died on the morning following this determination, yet Huth, whose blood-creatinin was 3.83 at the time that his clinical condition seemed to point to a fatal ending, recovered, showing ten days later practically normal blood figures. Thermic fever can be added therefore to the set of conditions which with high creatinin value, 5 mgm. or over, can be said to offer a fatal prognosis. For all intents and purposes these figures in thermic fever may be construed as similar in their interpretation to those obtained in severe uremic nephritis.

TREATMENT. A word or two in regard to the treatment of these cases: tub baths, gradually cooling the water to ice bath until the patient's temperature was reduced to 104° or under; stimulation with camphor, strychnin, and digitalin, free elimination, proctoclysis, with normal saline solution. In general, symptomatic treatment.

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## THE CLASSIFICATION OF THE CHRONIC HIGH BLOOD-PRESSURE CASES.

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THERE is no subject in medicine which, in the present age, has more vital interest to physicians than that of the chronic diseases of the circulatory system. Figures in all vital statistics have shown us that all affections of the circulatory and renal systems are definitely on the increase. "Arterial diseases of various kinds,

[10] Arch. Int. Med., 1915, xvi, 536-546.

atheroma, aneurysm, etc., caused 15,685 deaths in 1915, or 23.3 per 100,000. This rate, although somewhat lower than the corresponding ones for 1912 and 1913, is higher than that for 1911, and is very much higher than that for 1900, which was 6.1."[1] True, the average length of life has been considerably prolonged in the past twenty-five years, yet it is not due to a decrease in the incidence of these chronic diseases. We are not absolutely certain yet of the causes which are concerned in the increase of the chronic affections of the circulatory and renal systems, but our modern life, with its stress and strain, its mental concentration and overindulgence in protein food, must have some large part in the production of these diseases.

The study of the cardiovascular system has been greatly advanced by the introduction of instruments of precision which measure the blood-pressure, record the radial pulse, the venous pulse, the venous pressure, and record on photographic paper or film the electrical variations of the parts of the heart. Again, the introduction of various tests to measure the functional capacity of the kidneys has added immensely to our knowledge of various kidney diseases, and has been of great help in prognosis. To keep pace with the advances in these closely allied branches of medicine taxes the time and energy of any man. All that we can hope to do is to gather together a few of the generalizations and apply them as best we can to our daily work.

Blood-pressure as an aid to diagnosis has been badly used and much abused. Some have even expected the instrument to name the diagnosis, and when it, unlike Balaam's ass, was silent the instrument was blamed and discarded as worthless. Such an attitude is puerile. It is largely the result of taking only systolic pressure and neglecting the important diastolic and pulse-pressure. Happily this conception of blood-pressure is now undergoing a change, and we confidently expect to find that much help in diagnosis and prognosis will be given by careful blood-pressure estimations.

Five so-called phases have been described in the auscultatory blood-pressure phenomenon.[2] The systolic pressure is read at the beginning of the first phase and the diastolic, as I have shown,[3] is read at the sudden transition from the third to the fourth phase, or where the loud third sound suddenly becomes dulled. From there to the fifth phase, that is, no sound, is usually only from 4 to 6 mm. For those who find difficulty in determining the sudden transition of sound the fifth phase may be used. Hooker and Southworth[4] conclude from their observations that for clinical purposes the diastolic pressure may be taken at the disappearance of

[1] United States Mortality Statistics for 1915.
[2] Korotkov: Mitt. d. k. mil. med. Akad. zu St. Petersburg, 1905, xi, 365.
[3] Warfield, L. M.: Arch. Int. Med., 1912, x, 258.
[4] Arch. Int. Med., 1914, xiii, 384.

all sounds (the fifth phase). I have called attention, however, to the fact that in cases of aortic insufficiency there is no fifth phase, that is, the fourth phase or dull tone is heard over the uncompressed artery, hence one could not use the point of disappearance of sound to determine diastolic pressure. Must one then conclude that there is no diastolic pressure in aortic insufficiency? That would be absurd. One can easily obtain the diastolic pressure by taking the sudden transition of third into fourth phase. As a matter of fact, except in cases of aortic insufficiency, one may use the fifth phase to determine the diastolic pressure. It must, however, be remembered that there is an error of 4 to 6 mm. normally and up to 16 mm. in high-pressure cases.[5]

In an irregular heart, especially in the cases due to myocardial disease, it is quite impossible to determine the true diastolic pressure. One can only approximate it and say that the pulse-pressure is low or high. As a matter of fact the real systolic pressure cannot be determined. For this figure the place on the scale where most of the beats are heard may be taken for the average systolic pressure. No one can seriously maintain that he can measure the diastolic pressure under all circumstances.

By means of the auscultatory method of measuring blood-pressure we are able to determine irregularities of force in the heart beats more easily than by listening to the heart sounds. A pulsus alternans is readily made out. The irregular tones heard over the brachial artery in cases of irregular heart action have been called "tonal arrhythmias."[6]

There are three parts to every blood-pressure estimation: the systolic, the diastolic, and the pulse-pressure. These I have called the pressure picture.[7] To these should be added the pulse-rate in order to make the reading complete. Obviously, to say that the systolic pressure is 140, 160, or 200 mm. Hg. conveys to more knowledge of what the heart is doing than a count of red cells alone determines the kind of anemia. We have long known that a great variety of influences modifies the systolic pressure. Some of the influences are psychic and are entirely beyond our or the patient's control. Some apparently trivial circumstances, a chance remark made to the patient, an occurrence earlier in the day the memory of which flashed through his mind, may and does increase the systolic pressure 20 mm. or more above the average normal for the person. The systolic pressure measures the total work of the heart at the moment when it is tested. In a few minutes it may be higher or lower. A pathological or, rather, a compensatory high systolic pressure is always high, but the height is subject to great variation which makes it unwise to attribute a drop in pressure to any therapeutic measure.

[5] Warfield, L. M.: Jour. Am. Med. Assn., 1913, lxi, 1251.
[6] Goodman and Howell: Am. Jour. Med. Sc., 1911, cxlii, 321.
[7] Warfield, L. M.: Ibid., 1911, cxlviii, 880.

The diastolic pressure measures the peripheral resistance. It measures the work of the heart, the potential energy,* up to the moment of the opening of the aortic valves. It is the actual pressure in the aorta. The diastolic pressure is not very variable; it is not subject to the same influences which disturb the systolic pressure. It fluctuates, as a rule, within a small range. It is not affected by diet, by mental excitement, by subconscious psychic influences, to anything like the extent to which the systolic pressure is affected by the action of these factors. The diastolic pressure is determined by the tone in the arterioles and is under the control of the vasomotor sympathetic system. Any agent which causes chronic irritation of the whole vasomotor system produces increase in the peripheral resistance with consequent rise in the diastolic pressure. Any agent which acts to produce thickening of the walls of the arterioles, narrowing their lumina, produces the same effect.

Such states naturally result in increased work on the part of the heart, which, as a result, hypertrophies in the left ventricle. The increase in size and strength is a compensatory process in order to keep the tissues supplied with their requisite quota of blood. Conversely, paralysis of the vasomotor system produces fall of diastolic pressure which, if long continued, results in death.

The diastolic pressure then is of importance for the following reasons:

1. It measures peripheral resistance.
2. It is the measure of the tonus of the vasomotor system.
3. It is one of the points to determine pulse-pressure.
4. Pulse-pressure measures the actual driving force, the kinetic energy of the heart.
5. It enables us to judge of the volume output, for pulse-pressure (PP) × pulse-rate (PR) = volume output in most instances.
6. It is more stable than the systolic pressure, subject to fewer more or less unknown influences.
7. It is increased by exercise.
8. It is increased by conditions which increase peripheral resistance.
9. The gradual increase of diastolic pressure means harder work for the heart to supply the parts of the body with blood.
10. Increased diastolic pressure is always accompanied by increased pulse-pressure and increased size of the left ventricle, temporarily (exercise) or permanently.
11. Decreased diastolic pressure goes hand in hand with vasomotor relaxation, as in fevers, etc.
12. Low diastolic pressure is frequently pathognomonic of aortic insufficiency.
13. When the systolic and diastolic pressures approach, heart failure is imminent either when pressure picture is high or low.

* Stone, W. J.: Arch. Int. Med., 1915, xvi, 775.

Pulse-pressure measures the actual head of pressure which maintains the circulation, the force driving the blood to the periphery. As the circulatory bed widens the pulse-pressure diminishes until at the capillaries it is only about 5 mm. Hg.

When all these factors are taken into consideration it becomes apparent that the diastolic pressure is most important, if not the most important, part of the pressure picture. I am not unaware of the value of the great mass of statistical evidence which shows the bad prognostic import of high systolic pressure alone. One could show by the red cell count alone the seriousness of the low count, but one would fail to differentiate types of anemia and be unable to offer really valuable evidences of the gravity of anemia. So while conclusions drawn from statistics of high systolic pressure are in general correct, there is necessarily much that is incorrect and grossly misleading in them. I feel that only when we study our cases with the whole pressure pictures before us will we be able to draw really valuable conclusions for everyday use.

Figures for the normal pressure picture vary somewhat with sex and age. In adults we may safely assume that a systolic pressure which is constantly over 150 mm. Hg. is abnormal; a diastolic constantly over 90 is abnormal and a pulse-pressure over 50 mm. is an increased pressure.

Normally the pulse-pressure varies from 30 or under, low to 50 or above, high. We have found that in all of the cases of high pulse-pressure the left ventricle was dilated—it actually held more blood than a normal ventricle holds. The arch of the aorta was also dilated, easily revealed by auscultating over the manubrium,[9] and the seat of a definite sclerosis which rendered it less capable of expansion when the blood from the left ventricle was thrown into it at systole. The sequence of events conjecturally is as follows: There is arteriosclerosis of the aorta (usually the nodular), diffuse thickening of the walls of the large arteries, also some concomitant fibrous change in the myocardium. The elasticity of the aorta is reduced, hence the evenly distributed force which normally keeps the blood moving between systoles is absent. In order for the circulation to be carried on out to the extreme periphery, so that all the organs receive blood, more blood must be thrown at each systole into the aorta. The force also must be greater because the distributing tubes are no longer capable of doing their full share in maintaining the circulation in equilibrium. This not only causes hypertrophy of the left ventricle but actual increase of size of the cavity of the ventricle in order to accommodate the added amount of blood which must be put out at systole. As a result the left ventricle hypertrophies and dilates. The pathological process which is present in the myocardium by its very nature tends to increase gradually. More muscle fibers

[9] Warfield, L. M.: Jour. Am. Med. Ass'n., 1917, lxviii, 824.

are destroyed, more dilatation supervenes, more compensatory hypertrophy of the remaining fibers takes place. A vicious circle thus becomes established until finally the heart loses the power of carrying on the circulation and decompensation sets in. While we cannot prove this sequence absolutely, we believe that our clinical studies and our pathological studies make it more than probable that events occur in the order in which they have been related.

Again, the diastolic pressure, the peripheral resistance, does not rise in these cases above 110 mm. Usually it is between 90 and 100 mm. The systolic rarely goes above 200 mm. The pulse-pressure then may be from 80 to 110 mm. The larger the pulse-pressure the greater the cavity of the left ventricle and the more dilated is the arch of the aorta.

Stone[10] has divided the cases of hypertension into the cerebral and cardiac types. He finds there is a difference in prognosis and in mode of death in the two groups. He thinks that the pressure-ratio (heart load) which he has made use of will enable one to determine the type of case. I cannot say that I have found his method applicable except to a very few cases, although I agree with him in his contention that such a separation of cases is possible. I have felt the need of a better classification of the cases of chronic hypertension. For the past few years the cases have been critically studied, and it is believed that Stone's first group is composed of two groups, in general usually differentiable. I should therefore propose a classification into three groups. Syphilis is not an etiological factor in these cases. It is not contended that these groups are absolutely distinct. There are variations and combinations which render an exact separation into groups impossible. Bearing this in mind the following classification of the chronic high blood-pressure cases is submitted.

Group A. *Chronic Nephritis.* These are the cases with a high-pressure picture, that is to say, high systolic (200+) and high diastolic (120–140+). The pulse-pressure is much increased. The palpable arteries are hard and fibrous. There is puffiness of the under eyelids, which is more pronounced in the morning on arising. Polyuria with low specific gravity and nycturia are present. There are almost constant traces of albumin in the urine, with hyaline and finely granular casts.

Functionally these kidneys are much under normal. The functional capacity determined by Mosenthal's modification of the Schlayer-Hedinger method shows a marked inability to concentrate salts and nitrogen. The phthalein output is below normal. As the case advances the phthalein output becomes less and less until a period is reached when there are only traces or complete suppression

[10] Loc. cit.

at the end of a two-hour period. Such patients may live for ten weeks (one of our cases), all the time showing mild uremic symptoms, and suddenly pass into coma and die.

The natural end of patients in this group is either uremia or cardiac decompensation (so-called cardiorenal disease). Cerebral accidents may happen to a small number. It is only to this group, in my opinion, that the term cardiorenal disease should be applied. Formerly I believed that all high systolic pressure cases were cases of chronic nephritis of some definite degree. From the purely pathological stand-point that is true, but from the real, the functional stand-point, it is far from being the true state of the cases.

In this group there is marked hypertrophy and moderate dilatation of the left ventricle with dilatation and nodular sclerosis of the aorta. The kidneys are firm, red, small, coarsely granular, the cortex much reduced, the capsule adherent. Cysts are common. It is the familiar primary contracted kidney. Mallory calls this capsular-glomerulo-nephritis. The etiology is obscure. Often no cause can be found. Again, there is a history of some kidney involvement following one of the acute infectious diseases, or it may follow the nephritis of pregnancy. Usually, however, these cases fall into the group of secondary contracted kidneys, chronic parenchymatous nephritis.

Case I.—R. Z., a woman, aged thirty-six years, was seen July 26, 1916, in coma. There was a history of typhoid fever at nineteen years, but no other disease. She had had nine full-term pregnancies, the last one thirteen months previously. For a week before the onset of the present illness she had complained of severe headaches and dizziness. There were no heart symptoms. For the past year she has had nycturia. Physical examination revealed tubular breathing beneath the manubrium, a few rales in the chest, an enlarged heart (left side), with a systolic murmur over the aortic area. Blood-pressure was 178–125–53, the pulse-rate 96, leukocytes 27,250. Venesection of 500 c.c. of blood and intravenous injections of 500 c.c. of 5 per cent. $NaHCO_3$ in normal saline were employed. Lumbar puncture withdrew 60 c.c. of clear fluid under pressure with 6 cells per cubic millimeter. The eye-grounds showed distinct haziness of the disks and dilatation of the veins. Blood-pressure after venesection was 164–122–42, pulse 76, but in a few days rose to 222–142–80, pulse 70. A second venesection of 400 c.c. and proctoclysis of 1000 c.c. saline solution was tried. The blood-pressure now was 198–140–58. The pH of the blood was 7.6, the alkaline reserve was 35 volume per cent. (van Slyke), and the $CO_2$ tension of the alveolar air (Marriott) was 25 mm. The phthalein on the day following the second venesection was 45 per cent. in two hours. The urine at first showed 500 c.c. in twenty-four hours, specific gravity 1016, albumin and casts. Later she passed 1300 to 1600 c.c., with specific gravity around 1010. The

blood-pressure fluctuated considerably, reaching as low as 138–98–40, pulse 88. She was discharged improved September 10, 1916. At present (March, 1917) she is doing all her housework, but occasionally has headaches and attacks of dizziness.

GROUP B. This one might designate as the hereditary type, although there is not always a history in the antecedent. This group includes the robust, florid, exuberantly healthy people. They often are heard to boast that they have never had a doctor in their lives. They are usually thick-set or very large, fleshy people. The pressure picture is exceedingly high. The pulse-pressure is moderately increased. The arteries are rather large, fibrous, and often quite tortuous, although this is not always the case. Some persons have hard, small fibrous arteries. There is no puffiness beneath the eyes, no polyuria, and no nycturia as a rule. The urine is of normal amount, color, and specific gravity. Albumin is only rarely found and then in traces, but careful search of a centrifuged specimen invariably reveals a few hyaline casts. The phthalein excretion is normal or only slightly reduced. The kidneys excrete salt and nitrogen normally. It is in this group that apoplexy is found most frequently. The rupture of the vessel occurs when the victim is in perfect health, often without any warning. Occasionally when such a case recovers sufficiently to be around, cardiac decompensation sets in later and he dies then of the cardiac complications.

Pathologically the hearts of such persons are found to have the most enormous hypertrophy of the wall of the left ventricle. The cavity is somewhat enlarged, as is always the case when the pulse-pressure is increased, but the size of the cavity is not the striking feature. The aorta is fibrous, thick walled, and the arch is slightly dilated. There are patches of arteriosclerosis. One such case seen only at autopsy had a rupture of the aorta just above the sinus of Valsalva and died of hemopericardium. The kidneys are of normal size, dark red, firm, the capsule strips readily, the surface is smooth or finely granular, the cortex is not decreased. The pyramids are congested and red streaks extend into the cortex. Microscopically the capsules of the glomeruli are a trifle thickened; a few show hyaline changes. There is rather diffuse, mild, round-cell infiltration between the tubules. The tubular epithelium shows little or no demonstrable changes. The arterioles are generally the seat of a moderate thickening of the intima and media, but it is not usual to find obliterating endarteritis. There is evidently a diffuse fibrous change which has not affected either the tubules or glomeruli to any great extent.

CASE II.—L. C., a man, aged fifty-six years, stonemason by trade, is a stocky, thick neck individual. He has never been ill in his life until a year ago, when he fell from his chair unconscious. He had a right-sided hemiplegia which has cleared up so completely that except for a very slight drag to his foot he walks

perfectly well. He came in complaining of shortness of breath and cough. There was no swelling of the feet. Here evidently was left-heart decompensation. Examination showed the blood-pressure to be 240-130, pulse irregular, 104 to the minute. There was cyanosis and rales throughout both chests. The urine was normal in color, specific gravity 1025, small amount of albumin, few casts, hyaline and granular. The phthalein elimination was 65 per cent. in two hours. Under rest, purgatives, and digitalis he was much improved. He has since had two other apoplectic strokes, the last of which was fatal.

When these patients are seen with acute cardiac decompensation there is, of course, much albumin and many casts in the urine, and the phthalein output is, for the time being, decreased.

Group C. This might be called the arteriosclerotic high-tension group. The cases are usually over fifty years old. They are men and women who have lived high and thought hard. Often they have had periods of great mental strain. Many men in this group were athletes in their young manhood. Many have been fairly heavy drinkers, although never drinking to excess. They are usually well nourished and inclined to stoutness. The pressure picture is high systolic with normal or only slightly increased diastolic and large pulse-pressure. The arteries are large, full, fibrous, usually tortuous. The heart is very large, the apex far down and out. There is no polyuria; nycturia is uncommon, quite the exception. The urine is normal in color, amount, and specific gravity. Albumin is only rarely found and hyaline casts are not invariably present. The phthalein excretion is quite normal and the excretion of salt and nitrogen are also normal. The terminal condition in most of the patients in this group is cardiac decompensation. They may have several attacks from which they recover, but after every attack the succeeding one is produced by less exertion than the preceding one, and it becomes more and more difficult to control attacks. Eventually the patients become bed- or chair-ridden, and finally die of acute dilatation of the heart.

Occasionally patients in this group may have a cerebral attack, but in my experience this is uncommon. Pathologically the heart is large, at times true cor bovinum, dilated and hypertrophied. The cavity of the left ventricle is much dilated. The aorta is dilated and sclerosed.

The kidneys are increased in size, are firm, dark red in color, with fatty streaks in the cortex. The capsule strips readily and the cortex is normal in thickness or only slightly increased. The organ offers some resistance to the knife. The microscope shows small areas scattered throughout where the glomeruli are hyalinized, the stroma full of small round cells, the tubules dilated, and the cells are almost bare of protoplasm. Naturally the tubules are full of granular cast material. Also the arterioles show extensive intimal

thickening, fibrous in character, with occasional obliterating endarteritis. One gets the impression that the small sclerotic lesions are the result of anemia and gradual replacement of scattered glomeruli by fibrous tissue. For the most part the kidney, except for the chronic passive congestion, appears quite normal. One can readily understand that in such a kidney function could not have been interfered with.

Case III.—C. K., an active, stout, business man, aged fifty-six years, consulted me on account of shortness of breath and swelling of the feet in May, 1915. He had just returned from a hospital in another city, where he had gone with what was apparently cardiac decompensation. In his early manhood he had been a gymnast and was a prize winner. He has worked hard, often given way to violent paroxysms of temper, has eaten heavily but drunk very moderately. The heart was greatly enlarged, the arch of the aorta dilated, a mitral murmur was audible at the apex. The radials and temporals were large, tortuous, and fibrous. The blood-pressure picture ranged around 180–90–90. He was easily made dyspneic and had a tendency to swelling of the lower legs. The urine was acid, of normal specific gravity, normal in amount, normal phthalein, normal concentration of salt and nitrogen, contained albumin only when he was suffering from decompensation of the heart. Casts were always found. He finally died, after sixteen months, with all the symptoms of chronic myocardial insufficiency. The heart was enormous, a true cor bovinum. The kidneys were typical of this condition, possibly somewhat larger than usual.

The management of these groups of patients presents many difficulties. I believe that all these excessive pressures are compensatory. As has been said before, the systolic pressure is subject to considerable variations, but the diastolic remains fairly constant. Unless one can reduce the whole pressure picture therapy is useless. This reduction is far from being easy to accomplish. Occasionally one sees cases, such as two we have seen, in which there was uremia, high-pressure picture, almost total phthalein suppression. Eventually there was complete recovery, with normal pressure pictures. Such cases are the exceptions and are probably acutely toxic in origin. The chronic, slowly progressive cases do not act thus. The best we can do is to make searching inquiries into the mode of life and regulate it on a rational basis. All people of the three groups need regulation of habits and diet. We have not found drugs of value, with the possible exception of bichloride of mercury and potassium iodide. Even in the positive absence of syphilis these drugs in combination seem to exert a favorable influence on the factors which are causing the high pressure. Electricity in the form of the static current, the high-frequency current, and other forms are recommended. All aid in maintaining nutrition and hygiene, but in my experience have no permanent effect upon this class of cases which other measures do not have.

Naturally when decompensation sets in it is to be treated as it usually is in spite of the high systolic and high diastolic pressure. As a matter of fact I have seen the pressure picture reduced when compensation was restored under digitalis, and it is generally recognized that such is the case. Personally I believe that hydrotherapy, attention to the bowels, regular hours, great decrease in food, limitation of meats, especially purin base-containing meat products, substitution of buttermilk and cheese in the diet, are the chief means at our disposal for regulating the lives of those who fall into our hands before accidents have happened. But I realize that we have no means of actually preventing a cerebral hemorrhage, although we have some control over the development of a cardiac breakdown.

Finally, this grouping is of interest in respect to prognosis. Careful examination of the patient should enable us to predict with some degree of accuracy what will happen to him. We may be able to modify somewhat the course, and, in general, we can render a fairly correct prognosis. This is certainly of value to the patient's family as well as to the patient.

Conclusions. 1. In a blood-pressure reading the whole record should be taken, systolic, diastolic, pulse-pressure, and pulse-rate. The pressure picture is the term suggested for the figures representing the component parts of the blood-pressure reading.

2. The diastolic and pulse-pressure give us more information than the systolic pressure.

3. There are three groups of high-pressure cases, called (A) chronic interstitial nephritis, (B) hereditary or cerebral type, (C) arteriosclerotic or cardiac type. Causes of death are usually anemia in group A, cerebral hemorrhage in group B, and cardiac decompensation in group C.

4. The term cardiorenal disease should be reserved for the cases of group A, which suffer from cardiac decompensation; the term hypertensive cardiovascular disease for cases of group B. Myocardial insufficiency covers most of the cases in group C.

5. Prognosis is much more intelligently given when this grouping of cases is followed.